In the Claims:

1. (Original) A method for identifying a nucleic acid expressed in a concentration dependent manner, comprising:

determining a first nucleic acid expression profile of a first cell at a first position in an agent concentration gradient,

determining a second nucleic acid expression profile of a second cell at a second position in the agent concentration gradient,

determining a difference between the first and second nucleic acid expression profiles,

wherein the first position in the agent concentration gradient corresponds to a first concentration of agent, and the second position in the agent concentration gradient corresponds to a second concentration of agent, and at least the second cell has migrated through the agent concentration gradient.

2. (Original) The method of claim 1, wherein the nucleic acid expression profile is an mRNA expression profile.

3. (Original) The method of claim 1, wherein the agent concentration gradient is a ligand concentration gradient.

4. (Original) The method of claim 1, wherein the agent concentration gradient is a chemokine concentration gradient.

5. (Original) The method of claim 4, wherein the chemokine concentration gradient is selected from the group consisting of SDF-1α, SDF-1β, IP-10, MIG, GROα, GROβ, GROγ, IL-8, PF4, MCP, MIP-1α, MIP-1β, MIP-1γ (mouse), MCP-2, MCP-3, MCP-4, MCP-5 (mouse), RANTES, fractalkine, lymphotactin, CXC, IL-8, GCP-2, ENA-78, NAP-2, IP-10, MIG, I-TAC, SDF-1α, BCA-1, PF4, Bolekine, HCC-1, Leukotactin-1 (HCC-2, MIP-5), Eotaxin, Eotaxin-2 (MPIF2), Eotaxin-3 (TSC), MDC, TARC, SLC (Exodus-2, 6CKine), MIP-3α (LARC, Exodus-1), ELC (MIP-3β), I-309, DC-CK1 (PARC, AMAC-1), TECK, CTAK, MPIF1 (MIP-3), MIP-5 (HCC-2), HCC-4 (NCC-4), C-10 (mouse), C Lymphotactin, and CX_3C Fracktelkine (Neurotactin) and ITAC concentration gradients.

6. (Original) The method of claim 3, wherein the agent concentration gradient is a cytokine concentration gradient.

7. (Original) The method of claim 6, wherein the cytokine concentration gradient is selected from the group consisting of PAF, N-formylated peptides, C5a, LTB_4 and LXA_4, CXC, IL-8, GCP-2, GRO, GROα, GROβ, GROγ, ENA-78, NAP-2, IP-10, MIG, I-TAC, SDF-1α, BCA-1, PF4, Bolekine, MIP-1α, MIP-1β, RANTES, HCC-1, MCP-1, MCP-2, MCP-3, MCP-4, MCP-5 (mouse),

Leukotactin-1 (HCC-2, MIP-5), Eotaxin, Eotaxin-2 (MPIF2), Eotaxin-3 (TSC), MDC, TARC, SLC (Exodus-2, 6CKine), MIP-3α (LARC, Exodus-1), ELC (MIP-3β), I-309, DC-CK1 (PARC, AMAC-1), TECK, CTAK, MPIF1 (MIP-3), MIP-5 (HCC-2), HCC-4 (NCC-4), MIP-1γ (mouse), C-10 (mouse), C Lymphotactin, and CX_3C Fracktelkine (Neurotactin), SDF-1α, SDF-1β, met-SDF-1β, IL-1, IL-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-10, IL-12, IL-15, IL-18, TNF, IFN-α, IFN-β, IFN-γ, granulocyte-macrophage colony stimulating factor (GM-CSF), granulocyte colony stimulating factor (G-CSF), macrophage colony stimulating factor (M-CSF), TGF-β, FLT-3 ligand, VEGF, DMDA, endothelin and CD40 ligand concentration gradients.

8. (Original) The method of claim 1, wherein the first concentration of agent is a zero concentration of agent, and the second concentration of agent is a non-zero concentration of agent.

9. (Original) The method of claim 1, wherein the first concentration of agent is greater than the second concentration of agent.

10. (Original) The method of claim 1, wherein the first cell has migrated through the agent concentration gradient.

11. (Currently amended) The method of claim 1 ~~or 10~~, wherein migration through the agent concentration gradient is fugetactic migration.

12. (Currently amended) The method of claim 1 ~~or 10~~, wherein migration through the agent concentration gradient is chemotactic migration.

13. (Original) The method of claim 1, wherein the nucleic acid expression profile is determined by Northern analysis.

14. (Original) The method of claim 1, wherein the nucleic acid expression profile is determined by polymerase chain reaction (PCR) analysis.

15. (Original) The method of claim 1, wherein the nucleic acid expression profile is determined by nucleic acid chip analysis.

16. (Original) The method of claim 1, wherein the gradient is a step gradient.

17. (Original) The method of claim 1, wherein the gradient is a continuous gradient.

18. (Original) The method of claim 1, wherein the gradient comprises a second gradient co-existing with the first gradient.

19. (Original) The method of claim 1, wherein the first and second cells are adult cells.

20. (Original) The method of claim 1, wherein the first and second cells are human cells.

21. (Original) The method of claim 1, wherein the first and second cells are primary cells.

22. (Original) The method of claim 1, wherein the first and second cells are hemopoietic cells.

23. (Original) The method of claim 1, wherein the first and second cells are T lymphocytes.

24. (Original) The method of claim 1, wherein the first and second cells are neural cells.

25. (Original) A method for identifying a compound that can modulate cell migration in one or more agent concentration gradients comprising:

contacting a migratory cell in an agent concentration gradient with a test compound;

determining the nucleic acid expression profile in the cell; and

identifying a change in expression of a gene expression product.

26. (Original) The method of claim 25, wherein the cell migration is chemotaxic migration and the gene expression product is a chemotactic specific gene expression product.

27. (Original) The method of claim 25, wherein the cell migration is fugetaxic migration and the gene expression product is a chemotactic specific gene expression product.

28. (Original) The method of claim 25, wherein the nucleic acid expression profile is an mRNA expression profile.

29. (Original) The method of claim 25, wherein the agent concentration gradient is a ligand concentration gradient.

30. (Original) The method of claim 25, wherein the agent concentration gradient is a chemokine concentration gradient.

31. (Original) The method of claim 30, wherein the chemokine concentration gradient is selected from the group consisting of SDF-1α, SDF-1β, IP-10, MIG, GROα, GROβ, GROγ, IL-8, PF4, MCP, MIP-1α, MIP-1β, MIP-1γ (mouse), MCP-2, MCP-3, MCP-4, MCP-5 (mouse), RANTES, fractalkine, lymphotactin, CXC, IL-8, GCP-2, ENA-78, NAP-2, IP-10, MIG, I-TAC, SDF-1α, BCA-1, PF4, Bolekine, HCC-1, Leukotactin-1 (HCC-2, MIP-5), Eotaxin, Eotaxin-2 (MPIF2), Eotaxin-3 (TSC), MDC, TARC, SLC (Exodus-2, 6CKine), MIP-3α (LARC, Exodus-1), ELC (MIP-3β), I-309, DC-CK1 (PARC, AMAC-1), TECK, CTAK, MPIF1 (MIP-3), MIP-5 (HCC-2), HCC-4 (NCC-4), C-10 (mouse), C Lymphotactin, and CX_3C Fracktelkine (Neurotactin) and ITAC concentration gradients.

32. (Original) The method of claim 25, wherein the agent concentration gradient is a cytokine concentration gradient.

33. (Original) The method of claim 32, wherein the cytokine concentration gradient is selected from the group consisting of the cytokine concentration gradient is selected from the group consisting of PAF, N-formylated peptides, C5a, LTB_4 and LXA_4, CXC, IL-8, GCP-2, GRO, GROα, GROβ, GROγ, ENA-78, NAP-2, IP-10, MIG, I-TAC, SDF-1α, BCA-1, PF4, Bolekine, MIP-1α, MIP-1β, RANTES, HCC-1, MCP-1, MCP-2, MCP-3, MCP-4, MCP-5 (mouse), Leukotactin-1 (HCC-2,

MIP-5), Eotaxin, Eotaxin-2 (MPIF2), Eotaxin-3 (TSC), MDC, TARC, SLC (Exodus-2, 6CKine), MIP-3α (LARC, Exodus-1), ELC (MIP-3β), I-309, DC-CK1 (PARC, AMAC-1), TECK, CTAK, MPIF1 (MIP-3), MIP-5 (HCC-2), HCC-4 (NCC-4), MIP-1γ (mouse), C-10 (mouse), C Lymphotactin, and CX_3C Fracktelkine (Neurotactin), SDF-1α, SDF-1β, met-SDF-1β, IL-1, IL-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-10, IL-12, IL-15, IL-18, TNF, IFN-α, IFN-β, IFN-γ, granulocyte-macrophage colony stimulating factor (GM-CSF), granulocyte colony stimulating factor (G-CSF), macrophage colony stimulating factor (M-CSF), TGF-β, FLT-3 ligand, VEGF, DMDA, endothelin and CD40 ligand concentration gradients.

34. (Original) The method of claim 25, wherein the nucleic acid expression profile is determined at two different concentrations of agent.

35. (Original) The method of claim 34, wherein the two different concentrations of agent are a zero concentration of agent and a non-zero concentration of agent.

36. (Original) The method of claim 35, wherein the cell at a zero concentration of gradient has migrated through a gradient.

37. (Original) The method of claim 25, wherein the nucleic acid expression profile is determined by Northern analysis.

38. (Original) The method of claim 25, wherein the nucleic acid expression profile is determined by polymerase chain reaction (PCR) analysis.

39. (Original) The method of claim 25, wherein the nucleic acid expression profile is determined by nucleic acid chip analysis.

40. (Original) The method of claim 25, wherein the gradient is a step gradient.

41. (Original) The method of claim 25, wherein the gradient is a continuous gradient.

42. (Original) The method of claim 25, wherein the gradient comprises a second gradient co-existing with the first gradient.

43. (Original) The method of claim 25, wherein the cell is an adult cell.

44. (Original) The method of claim 25, wherein the cell is a human cell.

45. (Original) The method of claim 25, wherein the cell is a primary cell.

46. (Original) The method of claim 25, wherein the cell is a hemopoietic cell.

47. (Original) The method of claim 25, wherein the cell is a T lymphocyte.

48. (Original) The method of claim 25, wherein the cell is a neural cell.

49. (Original) A method for inhibiting cell fugetaxis comprising

contacting a cell undergoing or likely to undergo fugetaxis with an agent that inhibits a fugetaxis specific gene expression product in an amount effective to inhibit fugetaxis.

50. (Original) The method of claim 49, wherein the fugetaxis specific gene expression product is a nucleic acid or a peptide.

51. (Original) The method of claim 49, wherein the fugetaxis specific gene expression product is a signaling molecule.

52. (Original) The method of claim 49, wherein the signaling molecule is selected from the group consisting of cell division cycle 42, annexin A3, Rap1 guanine nucleotide exchange factor, adenylate cyclase 1, JAK binding protein, and Rho GDP dissociation inhibitor alpha.

53. (Original) The method of claim 52, wherein the signaling molecule is selected from the group consisting of cell division cycle 42 (cdc42), ribosomal protein S6 kinase, BAI1-associated protein 2, GTPase regulator associated with FAK, protein kinase C-beta 1, phosphoinositide-specific phospholipase C-beta 1, nitric oxide synthase 1, phosphatidylinositol-4-phosphate 5-kinase, and MAP kinase kinase kinase kinase 4.

54. (Original) The method of claim 49, wherein the fugetaxis specific gene expression product is a extracellular matrix related molecule.

55. (Original) The method of claim 54, wherein the extracellular matrix related molecule is selected from the group consisting of chitinase 3-like 1 (cartilage glycoprotein-39), carcinoembryonic antigen-related cell adhesion molecule 6, matrix metalloproteinase 8 (neutrophil collagenase), integrin cytoplasmic domain-associated protein 1, ficolin (collagenfibrinogen domain-containing) 1, epithelial V-like antigen 1, vascular endothelial growth factor (VEGF), fibulin 1, carcinoembryonic antigen-related cell adhesion molecule 3, and lysosomal-associated membrane protein 1.

56. (Original) The method of claim 49, wherein the fugetaxis specific gene expression product is a cytoskeleton related molecule.

57. (Original) The method of claim 56, wherein the cytoskeleton related molecule is selected from the group consisting of ankyrin 1 (erythrocytic), S100 calcium-binding protein A12 (calgranulin C), plectin 1 (intermediate filament binding protein, 500kD), microtubule -associated protein RPEB3, microtubule-associated protein 1A like protein (MILP), capping protein (actin filament, gelsolin-like), and ankyrin 2 (neuronal).

58. (Original) The method of claim 49, wherein the fugetaxis specific gene expression product is a cell cycle molecule.

59. (Original) The method of claim 58, wherein the cell cycle molecule is selected from the group consisting of v-kit Hardy-Zuckerman 4 feline sarcoma viral oncogene homolog, lipocalin 2 (oncogene 24p3), lectin, (galactoside-binding, galectin 3), RAB31 (member RAS oncogene family),

disabled (Drosophila) homolog 2 (mitogen-responsive phosphoprotein), RAB9 (member RAS oncogene family, pseudogene 1), and growth differentiation factor 8.

60. (Original) The method of claim 49, wherein the fugetaxis specific gene expression product is an immune response related molecule.

61. (Original) The method of claim 61, wherein the immune response related molecule is selected from the group consisting of major histocompatibility complex (class II, DR alpha), S100 calcium-binding protein A8 (calgranulin A), small inducible cytokine subfamily A (Cys -Cys), eukaryotic translation initiation factor 5A, small inducible cytokine subfamily B (Cys-X-Cys) (member 6, granulocyte chemotactic protein 2), Fc fragment of IgG binding protein, CD24 antigen (small cell lung carcinoma cluster 4 antigen), MHC class II transactivator, T cell receptor (alpha chain), T cell activation (increased late expression), MKP-1 like protein tyrosine phosphatase, T cell receptor gamma constant 2, T cell receptor gamma locus, cytochrome P450 (subfamily IVF, polypeptide 3, leukotriene B4 omega hydroxylase).

62. (Original) The method of claim 49, wherein the cell is an immune cell.

63. (Original) The method of claim 49, wherein the contacting occurs in vivo in a subject having or at risk of having an abnormal immune response.

64. (Original) The method of claim 49, wherein the cell is a neural cell.

65. (Original) The method of claim 49, wherein the fugetaxis specific gene expression product is chemokine (C-X3-C) receptor 1.

66. (Original) A method for inhibiting cell chemotaxis comprising

contacting a cell undergoing or likely to undergo chemotaxis with an agent that inhibits a chemotaxis specific gene expression product in an amount effective to inhibit chemotaxis.

67. (Original) The method of claim 66, wherein the chemotaxis specific gene expression product is a nucleic acid or a peptide.

68. (Original) The method of claim 66, wherein the cell is a immune cell.

69. (Original) The method of claim 66, wherein the contacting occurs in vivo in a subject having or at risk of having an abnormal immune response.

70. (Original) The method of claim 66, wherein the abnormal immune response is an inflammatory response.

71. (Original) The method of claim 66, wherein the abnormal immune response is an autoimmune response.

72. (Original) The method of claim 71, wherein the autoimmune response is selected from the group consisting of rheumatoid arthritis, Crohn's disease, multiple sclerosis, systemic lupus erythematosus (SLE), autoimmune encephalomyelitis, myasthenia gravis (MG), Hashimoto's

thyroiditis, Goodpasture's syndrome, pemphigus (e.g., pemphigus vulgaris), Grave's disease, autoimmune hemolytic anemia, autoimmune thrombocytopenic purpura, scleroderma with anti-collagen antibodies, mixed connective tissue disease, polymyositis, pernicious anemia, idiopathic Addison's disease, autoimmune-associated infertility, glomerulonephritis (e.g., crescentic glomerulonephritis, proliferative glomerulonephritis), bullous pemphigoid, Sjögren's syndrome, insulin resistance, and autoimmune diabetes mellitus.

73. (Original) The method of claim 66, wherein the abnormal immune response is a graft versus host response.

74. (Original) The method of claim 66, wherein the chemotaxis specific gene expression product is a signaling molecule.

75. (Original) The method of claim 74, wherein the signaling molecule is selected from the group consisting of G protein-coupled receptor kinase 6, vaccinia related kinase 1, PTK2 protein tyrosine kinase 2, STAM-like protein containing SH3 and ITAM domains 2, signal-induced proliferation-associated gene 1, CD47 antigen (Rh-related antigen, integrin-associated signal transducer), and protein tyrosine phosphatase (non-receptor type 12).

76. (Original) The method of claim 74, wherein the signaling molecule is selected from the group consisting of PTK2 (focal adhesion kinase), MAP kinase kinase kinase kinase 2, guanine nucleotide binding protein, PT phosphatase (receptor), cdc42-binding protein kinase beta, Ral GEF (RalGPS1A), MAP kinase 7, autotaxin, inositol 1,4,5-triphosphate receptor, phosphoinositide-3-kinase gamma, PT phosphatase (non-receptor), RAS p21 protein activator (GAP), RAS guanyl releasing protein 2, and Arp23 complex 20kDa subunit.

77. (Original) The method of claim 66, wherein the chemotaxis specific gene expression product is a extracellular matrix related molecule.

78. (Original) The method of claim 77, wherein the extracellular matrix related molecule is selected from the group consisting of spondin 1 (f-spondin, extracellular matrix protein), collagen type XVIII (alpha 1), CD31 adhesion molecule, tetraspan 3, glycoprotein A33, and angio-associated migratory cell protein.

79. (Original) The method of claim 66, wherein the chemotaxis specific gene expression product is a cytoskeleton related molecule.

80. (Original) The method of claim 79, wherein the cytoskeleton related molecule is selected from the group consisting of actin related protein 23 complex (subunit 4, 20 kD), tropomyosin 2 (beta), SWISNF related matrix associated actin dependent regulator of chromatin (subfamily a, member 5), spectrin beta (non-erythrocytic 1), myosin light polypeptide 5 (regulatory), keratin 1, plakophilin 4, and capping protein (actin filament, muscle Z-line, alpha 2).

81. (Original) The method of claim 66, wherein the chemotaxis specific gene expression product is a cell cycle molecule.

82. (Original) The method of claim 81, wherein the cell cycle molecule is selected from the group consisting of FGF receptor activating protein 1, v-maf musculoaponeurotic fibrosarcoma (avian) oncogene homolog, cyclin-dependent kinase (CDC2-like) 10, TGFB inducible early growth response 2, retinoic acid receptor alpha, anaphase promoting complex subunit 10, RAS p21 protein activator (GTPase activating protein, 3-Ins-1,3,4,5, -P4 binding protein), cell division cycle 27, programmed cell death 2, c-myc binding protein, mitogen-activated protein kinase kinase kinase 1, TGF beta receptor III (betaglycan, 300 kDa), and G1 to S phase transition 1.

83. (Original) The method of claim 66, wherein the chemotaxis specific gene expression product is an immune response related molecule.

84. (Original) The method of claim 83, wherein the immune response related molecule is selected from the group consisting of major histocompatibility complex class II DQ beta 1, bone marrow stromal cell antigen 2, Burkitt lymphoma receptor 1 (GTP binding protein, CXCR5), CD7 antigen (p41), Stat2 type a, T cell immune regulator 1, and interleukin 21 receptor.

85. (Original) The method of claim 66, wherein the cell is a neural cell.

86. (Original) A method for promoting cell fugetaxis comprising

contacting a cell with a non-chemokine agent that promotes fugetaxis in an amount effective to promote fugetaxis.

87. (Original) The method of claim 86, wherein the contacting occurs in vivo in a subject having a disorder characterized by lack of fugetaxis.

88. (Original) The method of claim 86, wherein the cell is a hematopoietic cell.

89. (Original) The method of claim 88, wherein the hematopoietic cell is a T lymphocyte.

90. (Original) The method of claim 86, wherein the cell is a neural cell.

91. (Original) A method for promoting cell chemotaxis comprising

contacting a cell with a non-chemokine agent that promotes chemotaxis in an amount effective to promote chemotaxis.

92. (Original) The method of claim 91, wherein the contacting occurs in vivo in a subject having a disorder characterized by lack of chemotaxis.

93. (Original) The method of claim 91, wherein the cell is a hematopoietic cell.

94. (Original) The method of claim 93, wherein the hematopoietic cell is a T lymphocyte.

95. (Original) The method of claim 91, wherein the cell is a neural cell.

96. (Currently amended) The method of claim 62 ~~or 68~~, wherein the immune cell is a selected from the group consisting of T-cells, B-cells, NK cells, dendritic cells, monocytes and macrophages.

97. (Original) The method of claim 96, wherein the immune cell is an inflammatory selected from the group consisting of neutrophils, basophils, eosinophils and mast cells.

98. (Currently amended) The method of claim 88 ~~and 93~~, wherein the hematopoietic cell is an immune cell is a selected from the group consisting of T-cells, B-cells, NK cells, dendritic cells, monocytes and macrophages.

99. (Original) The method of claim 98, wherein the immune cell is an inflammatory selected from the group consisting of neutrophils, basophils, eosinophils and mast cells.

100. (Original) A method for promoting neutrophil chemotaxis comprising contacting a neutrophil with IL-8 in an amount effective to promote chemotaxis.

101. (Original) The method of claim 100, wherein the neutrophil is contacted with a low concentration of IL-8.

102. (Original) The method of claim 101, wherein the low concentration of IL-8 is between about 10 ng/ml to about 500 ng/ml.

103. (Original) The method of claim 100, wherein the contacting occurs in vivo in a subject having a disorder characterized by lack of neutrophil chemotaxis.

104. (Original) The method of claim 103, wherein the disorder is selected from the group consisting of bacterial infections and granulomatous diseases.

105. (Original) The method of claim 103, wherein IL-8 is provided to the subject on a material surface coated with IL-8.

106. (Original) The method of claim 105, wherein the material surface is implanted within the subject.

107. (Original) The method of claim 103, wherein IL-8 is provided to the subject in a controlled release formulation.

108. (Original) A method for promoting neutrophil fugetaxis comprising contacting a neutrophil with IL-8 in an amount effective to promote fugetaxis.

109. (Original) The method of claim 108, wherein the neutrophil is contacted wih a high concentration of IL-8.

110. (Original) The method of claim 109, wherein the concentration of IL-8 is between about 1 microgram/ml to about 10 micrograms/ml.

111. (Original) The method of claim 108, wherein the contacting occurs in vivo in a subject having a disorder characterized by lack of neutrophil fugetaxis.

112. (Original) The method of claim 111, wherein the disorder is selected from the group consisting of inflammatory or immune mediated diseases, rejection of a transplanted organ or tissue, rheumatoid arthritis, automimmune diseases and asthma.

113. (Original) The method of claim 108, wherein IL-8 is provided to the subject on a material surface coated with IL-8.

114. (Original) The method of claim 113, wherein the material surface is implanted within the subject.

115. (Original) The method of claim 108, wherein IL-8 is provided to the subject in a controlled release formulation.

116. (Original) A method for inhibiting IL-8 induced neutrophil chemotaxis comprising contacting a neutrophil with wortmannin in an amount effective to inhibit chemotaxis and optionally induce fugetaxis by the neutrophil.

117. (Original) A method for inhibiting IL-8 induced neutrophil fugetaxis comprising contacting a neutrophil with LY294002 in an amount effective to inhibit fugetaxis and optionally induce chemotaxis by the neutrophil.

118. (Currently amended) The method of claim 4 ~~or 30~~, wherein the chemokine concentration gradient is a SDF-1 concentration gradient.

119. (Currently amended) The method of claim 4 ~~or 30~~, wherein the chemokine concentration gradient is an IL-8 concentration gradient.

120. (Currently amended) The method of claim 6 ~~or 32~~, wherein the cytokine concentration gradient is a SDF-1 concentration gradient.

121. (Currently amended) The method of claim 6 ~~or 32~~, wherein the cytokine concentration gradient is an IL-8 concentration gradient.